pulmonary area. Contrary to what is usually the case in congenital heart lesions, cyanosis and clubbing of the fingers do not occur.

Five cases have come under Gibson's observation during the last few years. One of these he reports in detail, to substantiate his statement that cases of uncomplicated patent ductus arteriosus can be readily diagnosed. The main points in this case were as follows: The patient was a woman, aged thirty-one years. There was nothing of importance in the family history. She had always been subject to palpitation on exertion or excitement. There was no clubbing of the fingers, but a slight bluish color to the lips. The pulse was usually regular and equal in force and rhythm. There was slight bulging of the second, third, and fourth left costal cartilages, especially the second, close to the sternum. The point of maximum cardiac impulse was in the fifth interspace three and a half inches from the sternum. The right border of the heart reached one and a half inches and the left four and a half inches from the midsternum. There was a thrill over the base of the heart. It was felt with greatest intensity in the third left intercostal space, two and a half inches from the midsternum. It began shortly after the apical impulse and continued almost to the next apex beat. The shock of the pulmonary second sound could be distinctly felt during its continuance. At the base of the heart an almost continuous rushing murmur, beginning shortly after the first sound, which was clear at its commencement, and continuing almost to the beginning of the next first sound, was heard. It had its maximum intensity in the third left interspace, about two and a half inches from the midsternal line, but was heard all over the base of the heart. The second sound was heard distinctly in the middle of this murmur; it was distinctly accentuated and sometimes doubled. The sounds at the other areas were all audible, some being more obscured than others by the transmitted murmur.

With a thrill and murmur beginning after the first sound and continuing until after the end of the second sound, there was no possibility entertained in Gibson's mind that the physical signs were due to an alveolar lesion. It seemed to him that no other diagnosis was possible than patency of the ductus arteriosus.

The patient at a subsequent date was admitted with acute lobar pneumonia, from which she eventually died. An autopsy was obtained and Gibson's diagnosis of patent arterial duct was substantiated. The ductus was so short that at first sight it seemed as if the left pulmonary artery was actually adherent to the aorta. The duct admitted a No. 12 to 14 bougie. There were no abnormal communications between the various heart cavities. The various valves were normal; the tricuspid orifice was dilated, however. The right ventricle was dilated and hypertrophied, the wall measuring three-sixteenths of an inch in thickness.

Gibson states that this case shows that in certain instances the diagnosis must in large part be made "from the stand-point of clinical observation, instead of from the consideration of the pathological changes in the heart and the resulting mechanical disturbances."

The writer sums up the essential facts upon which the diagnosis of persistent ductus arteriosus may be made with perfect confidence as follows:

occurring paroxysmally without albuminuria. Ebstein discusses the colic, with critical reference to the literature, showing the uncertain relation of gallstone colic and glycosuria, the difficulties in the diagnosis of pancreatic colic, and suggesting that in his own case the colic may have been one of the not unusual visceral neuralgias of diabetes. He concluded that uncomplicated biliary colic cannot cause glycosuria.

As regards the œdema, in the case reported this was characterized by the rapidity with which it spread over the lower extremities and external genitals, less intensely in the upper part of the body and the face, and the quickness with which it disappeared, especially under the influence of diuretic and diaphoretic treatment. The examination of the heart gave no explanation of the œdema; there was no congestion, no cachexia, nor hydræmia. After the œdema subsided there was an erythematous flush on the skin and a feeling of soreness. The author was, therefore, led to think of an œdema due to nervous influence, perhaps by the action of a toxic substance.

Chemical Changes in the Blood in Pernicious Anæmia.—ERBEN (*Zeitschrift fur klin. Med.*, Band xl., p. 266) reports some investigations made in a case of pernicious anæmia. He found: 1. That the albumin is diminished, due to a decrease of the serum-albumin and the great reduction (to one-fourth of the normal by weight) of the red corpuscles. The individual red cells are hypertrophied and their albumin increased. 2. Among the various albuminous bodies the fibrin is absolutely diminished, but relatively normal, the albumin almost normal, the serum-globulin much reduced, so that almost all the loss is due to it. The author suggests that this loss is related to the progressive diminution of the red corpuscles, the appearance of derivatives of the hæmoglobin, the relative increase of lecithin, and the frequently associated atrophy of the intestinal mucosa. There may, indeed, be a causal relation in the failure of the organism, or the intestinal mucosa, to form globulins. 3. The water is increased, both in the serum and erythrocytes. The hypertrophic red cells are swollen. 4. Fat is present in normal amount, cholesterin greatly diminished, though the latter is still present in the red corpuscles in proportion to their organic matter. Lecithin is diminished in the serum, but increased in the red cells. 5. The watery extractives are diminished in the whole blood and the serum, the alcoholic extractives increased in both; but in the red cells both extractives are increased. 6. The ash is increased. 7. In the ash Na_2O and Cl are increased (no doubt as the result of hydræmia); CaO and MgO increased (as the result of destruction of bone, according to the author), K_2O and P_2O_5 lessened, though the red corpuscles contain more of the latter, the serum more of the former than normal. Iron is lessened, on the whole, though the serum contains it and the red cells show an excess. From the relations of the iron to the hæmoglobin it seems that in pernicious anæmia either the hæmoglobin is richer in iron than normal or the red cells contain iron in some other form. Perhaps both possibilities are true.

Post typhoid Suppuration of the Thyroid Gland.—SCHUDMAK and VLACHOS (*Wiener klin. Wochenschrift*, 1900, No. 29) report the case of a